Journal of the American College of Cardiology Vol. 51, No. 23, 2008

Published by Elsevier Inc.
ISSN 0735-1097/08/$34.00
doi:10.1016/j.jacc.2007.12.061

Ethnic Differences in Left Ventricular Remodeling in Highly-Trained Athletes

Relevance to Differentiating Physiologic Left Ventricular Hypertrophy From Hypertrophic Cardiomyopathy

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Objectives The purpose of this study was to evaluate ethnic differences in left ventricular (LV) remodeling between highly-trained athletes of African/Afro-Caribbean (black) and Caucasian (white) athletes.

Background The upper limits of left ventricular hypertrophy (LVH) are established in white athletes and aid the differentiation of physiologic LVH from hypertrophic cardiomyopathy (HCM). However, there are few data regarding LV remodeling in black athletes, in whom deaths from HCM are more prevalent.

Methods Between 2003 and 2007, 300 nationally ranked black male athletes (mean age 20.5 years) underwent 12-lead electrocardiogram and 2-dimensional echocardiography. The results were compared with 150 black and white sedentary individuals and 300 highly-trained white male athletes matched for age, size, and sport.

Results Black athletes exhibited greater LV wall thickness and cavity size compared with sedentary black and white individuals. Black athletes had greater LV wall thickness compared with white athletes (11.3 ± 1.6 mm vs. 10 ± 1.5 mm; $p < 0.001$). In absolute terms, 54 black athletes (18%) had LV wall thickness >12 mm compared with 12 white athletes (4%), and 3% of black athletes exhibited LV wall thickness ≥15 mm compared with none of the white athletes. Black athletes with LVH displayed an enlarged LV cavity and normal diastolic function.

Conclusions Black athletes develop a greater magnitude of LVH compared with white athletes; therefore, extrapolation of conclusions derived from white athletes has the potential of generating false-positive diagnoses of HCM in black athletes. (J Am Coll Cardiol 2008;51:2256–62) © 2008 by the American College of Cardiology Foundation

Participation in intensive physical exercise is associated with increased left ventricular (LV) dimensions (1–3). The upper limits of LV wall thickness are established in Caucasian (white) athletes (1,3) and serve an important role in differentiating physiologic LV hypertrophy (LVH) from hypertrophic cardiomyopathy (HCM) (4). There are few studies addressing cardiovascular adaptation in athletes of African/Afro-Caribbean origin (black), in whom deaths attributed to HCM are more common (5). It is uncertain whether extrapolation of conclusions relating to LV wall thickness measurements derived from white athletes can be applied accurately to confirm or refute the presence of HCM in black athletes. However, a study of intercollegiate black athletes suggested that black athletes are capable of developing substantial LVH (6), indicating that increases in pre-load and after-load associated with intensive exercise may be associated with a greater degree of LVH in black athletes compared with white athletes. Paucity of data on cardiovascular adaptation in black athletes has the potential of generating false-positive diagnoses of HCM and unnecessary disqualification from competitive sport. The present study aimed to evaluate ethnic differences in cardiovascular adaptation to intensive exercise between black and white athletes.

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Methods

Setting. The United Kingdom does not have a formal government-sponsored pre-participation screening program

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Manuscript received October 23, 2007; revised manuscript received December 12, 2007, accepted December 17, 2007.

for athletes; however, some national sporting organizations fund independent cardiovascular screening for recruits before acceptance for competition. Our organization has been responsible for evaluating athletes from the British Lawn Tennis Association, Premiership Soccer and Rugby League, and national swimming and rowing squads since 1996. In 2003, the program was extended to include basketball, boxing, and track events to enable assessment of elite black athletes.

Definitions. The term "black" was used to denote individuals of African or Afro-Caribbean descent (7,8). The ethnicity assigned to each black athlete and control relied purely on information provided on health questionnaires.

The term "elite athlete" was applied to individuals who underwent organized training and participated in a team or individual sport at a national level. The term "sedentary" was ascribed to individuals participating in <2 h of organized physical activity per week. Based on previous studies in athletes, "LVH" was defined as an end-diastolic LV wall thickness >12 mm (1,3).

Athletes. Between 2003 and August 2007, 300 asymptomatic elite black male athletes underwent 12-lead electrocardiogram (ECG) and 2-dimensional echocardiography during the peak competitive season. Athletes had a mean age of 20.5 ± 5.8 years (range 14 to 35 years) and a mean body surface area (BSA) of 1.94 ± 0.16 m^2 (range 1.3 to 2.29 m^2). Athletes were Afro-Caribbean (42%), West African (40%), or East African (18%) in origin. Athletes of East African origin had a lower BSA compared with other black athletes (1.89 ± 0.15 m^2 vs. 1.97 ± 0.16 m^2; $p < 0.001$). Athletes trained for an average of 14 ± 7 h (range 10 to 46 h) per week, and all had competed at the national level in the 4-year period. The athletes participated in 6 different sporting disciplines (soccer, n = 90 [30%]; boxing, n = 60 [20%]; basketball, n = 55 [18%]; track sprinting, n = 21 [7%]; long-distance running, n = 39 [13%]; rugby, n = 29 [10%]; and tennis, n = 6 [2%]). Written consent for evaluation was obtained from individuals ≥16 years and from a parent/guardian for those <16 years of age.

Control subjects. Control subjects comprised 150 black and 150 white sedentary males and 300 white male elite athletes of similar age and BSA as the study subjects. Sedentary control subjects were recruited from large secondary schools and community centers. Elite white athletes were recruited from the same sporting disciplines/clubs that provided black athletes for the study and therefore endured similar training programs.

Ethical approval for the study was obtained from the University Hospital Lewisham Research Ethics Committee.

Electrocardiography. A standard 12-lead ECG was recorded using a Marquette Hellige recorder (Milwaukee, Wisconsin) with a paper speed of 25 mm/s and amplification of 0.1 mV/mm (9). The Sokolow-Lyon voltage criteria were used to identify LVH (10).

Echocardiography. Two-dimensional echocardiography was performed by 2 experienced sonographers using GE Vivid I laptop portable (General Electric, Tirat Carmel, Israel) and Philips Sonos 7500 (Philips, Bothell, Washington) echocardiography machines. Images of the heart were obtained in the standard views as previously described (11). The LV wall thickness was measured from 2-dimensional long- and short-axis views in end-diastole at the level of the mitral valve leaflet tips and the papillary muscles; the greatest measurement defined maximal LV wall thickness. M-mode echocardiograms derived from 2-dimensional images in the parasternal long axis were used to measure LV end-diastolic and -systolic dimensions and the left atrial and aortic root diameters according to the American Society of Echocardiography (12). Repeat measurements were performed by a cardiologist blind to the identity of the subjects. Relative wall thickness (h/R), where h is the sum of the interventricular septal and posterior wall thickness in end-diastole (mm) and R is the LV cavity size in end-diastole (mm), was calculated for each athlete. Percentage LV shortening fraction was calculated as an index of systolic function. Pulsed Doppler recordings were performed at the distal margins of mitral valve leaflets for early (E) and late (A) diastolic velocities (13). Tissue Doppler imaging (TDI) of septal and lateral mitral annulular movement was obtained from the apical 4-chamber views, as previously described (14), to obtain early (E′) and late (A′) diastolic peak velocities. The ratio of early to late annular velocity (E′/A′) and the ratio of transmitral flow velocity to annular velocity (E/E′) were calculated to provide indexes of diastolic function. Left ventricular mass was calculated using the formula of Devereux (15).

Abbreviations and Acronyms

BP = blood pressure
BSA = body surface area
ECG = electrocardiography/electrocardiogram
HCM = hypertrophic cardiomyopathy
LV = left ventricular
LVH = left ventricular hypertrophy
TDI = tissue Doppler imaging

Athletes with LVH underwent upright exercise stress testing and 48-h Holter monitoring to check for the broader phenotype of HCM.

Exercise stress testing. An upright stress test was performed using the standard Bruce Protocol (16). The ECG and blood pressure (BP) were recorded at 1-min intervals. Athletes were exercised to volitional exhaustion and assessed specifically for the development of ischemic changes, abnormal or flat BP response (17), and tachyarrhythmias.

48-h ECG monitoring. The 48-h ambulatory ECG monitoring was performed to check specifically for supraventricular and/or ventricular tachyarrhythmias. Athletes were encouraged to continue usual day-to-day life activities, including exercise, during the investigation.

Statistical analysis. Data are expressed as mean ± SD. Statistical analyses were performed using the unpaired Student t test, univariate analysis of variance test with post hoc Bonferroni correction, and chi-square test. A stepwise multivariate linear model was produced to assess the rela-

Table 1 Comparison Between Black Athletes, White Athletes, and Black Control Subjects

	Black Athletes (n = 300)	White Athletes (n = 300)	Black Control Subjects (n = 150)	p Value (ANOVA)
Age (yrs)	20.5 ± 5.8 (14–35)	20.2 ± 4.9 (14–35)	19.4 ± 6.4 (14–35)	NS
BSA (m^2)	1.93 ± 0.2	1.89 ± 0.3	1.87 ± 0.5	NS
Systolic BP (mm Hg)	118 ± 7 (80–140)	115 ± 6 (95–120)	119 ± 12 (85–145)	NS
$LVWT_d$ (mm)	11.3 ± 1.6* (8–16)	10.0 ± 1.5 (7–14)	9.0 ± 1.2 (6–12)	<0.001
$LVID_d$ (mm)	53 ± 4.4† (44–64)	53.6 ± 4.1 (42–66)	49 ± 5.2 (28–55)	<0.001
LA diam. (mm)	36 ± 4† (20–48)	36 ± 4 (24–47)	31 ± 4 (23–41)	<0.001
h/R	0.42 ± 0.07*	0.36 ± 0.06	0.35 ± 0.06	<0.001
LVM (g)	286 ± 78* (113–618)	250 ± 62 (113–489)	225 ± 60 (110–360)	<0.001
E (m/s)	0.87 ± 0.2† (0.5–1.5)	0.89 ± 0.3 (0.5–1.2)	0.8 ± 0.2 (0.5–1.0)	NS
A (m/s)	0.4 ± 0.2 (0.2–0.7)	0.5 ± 0.3 (0.3–0.9)	0.5 ± 0.2 (0.3–0.8)	NS
E/A	2.3 ± 0.9 (1.6–4.8)	2.2 ± 1 (1.4–3.6)	1.3 ± 0.8 (1.2–3.5)	NS
E′ (m/s)	0.22 ± 0.2† (0.16–0.29)	0.23 ± 0.1 (0.17–0.29)	0.16 ± 0.01 (0.13–0.19)	<0.001
A′ (m/s)	0.05 ± 0.01† (0.03–0.08)	0.05 ± 0.01 (0.01–0.09)	0.07 ± 0.01 (0.04–0.09)	<0.001
E′/A′	2 ± 0.4† (1.7–4.6)	2 ± 0.5 (1.6–4.7)	1.6 ± 0.23 (1.1–2.4)	<0.001
E/E′	4.6 ± 1.3† (3.2–7.5)	4.5 ± 1.4 (3.4–7.2)	5.1 ± 1.2 (4.1–9.7)	<0.001
FS	35.2 ± 6.8 (20–51)	36.3 ± 5.7 (24–42)	36.1 ± 6.2 (22–50)	NS

Data expressed as mean ± standard deviation (range). *More significant compared with black control subjects and white athletes. †More significant compared with black control subjects but not with white athletes.

A = late mitral valve inflow peak velocity; A′ = late annular diastolic peak velocity; ANOVA = analysis of variance; BP = blood pressure; BSA = body surface area; E = early diastolic mitral valve peak inflow velocity; E′ = early diastolic annular peak velocity; FS = fractional shortening; h/R = relative wall thickness; LA diam. = left atrial diameter; $LVID_d$ = maximal left ventricular cavity dimension in end-diastole; LVM = left ventricular mass; $LVWT_d$ = maximal left ventricular wall thickness in end-diastole.

tionship between LV wall thickness as a dependent variable and weight, height, age, ethnicity, type of sport, and duration of training as independent variables. A 2-tailed p value of <0.05 was considered to be statistically significant. Intraobserver and interobserver reliability for maximal left ventricular wall thickness measurements was expressed as the coefficient of variance (%).

Results

Sedentary control subjects. Black and white control subjects were of similar age (19.4 ± 6.4 years vs. 20.5 ± 5.8 years) and BSA (1.89 ± 0.5 m^2 vs. 1.82 ± 0.2 m^2). There were no differences in LV wall thickness or LV cavity between black and white sedentary control subjects (9.0 ± 1.2 mm vs. 8.8 ± 1.3 mm and 49 ± 5.2 mm vs. 48 ± 4.0 mm, respectively). None of the control subjects exhibited a LV wall thickness >12 mm or LV cavity size >55 mm.

Cardiac dimensions in black athletes versus black control subjects and white athletes. Black athletes and white athletes exhibited a greater LV wall thickness, cavity size, and left atrial diameter compared with black control subjects. Both black athletes and white athletes displayed enhanced diastolic LV filling (E′ and E/E′) compared with black control subjects on TDI (Table 1). Black athletes demonstrated a greater magnitude of LV wall thickness and LV mass compared with white athletes, amounting to a 12% and 13% difference, respectively (Table 1). There were no differences in the LV cavity size, left atrial diameter, baseline LV function, and BP between black athletes and white athletes.

Black athletes with LVH. In absolute terms, 54 black athletes (18%) had LV wall thickness >12 mm (LVH) compared with 12 white athletes (4%) ($p < 0.001$). The maximal LV wall thickness measured in any black athlete was 16 mm, compared with 14 mm in any white athlete (Fig. 1). Nine black athletes (3%) exhibited substantial LV wall thickness ≥15 mm (Table 2).

Demographics. Black athletes with LVH had a larger BSA compared with black athletes without LVH (2.0 ± 0.2 m^2 vs. 1.92 ± 0.2 m^2; $p < 0.01$). There were no differences between the 2 groups in relation to age or intensity of training, but all black athletes with LVH were age ≥16 years. In terms of ancestral origin of black athletes, 50 (20%) of the 246 athletes of West African ancestry (including Caribbean individuals) exhibited LVH versus 4 (7%) of 54 athletes of East African origin ($p < 0.01$).

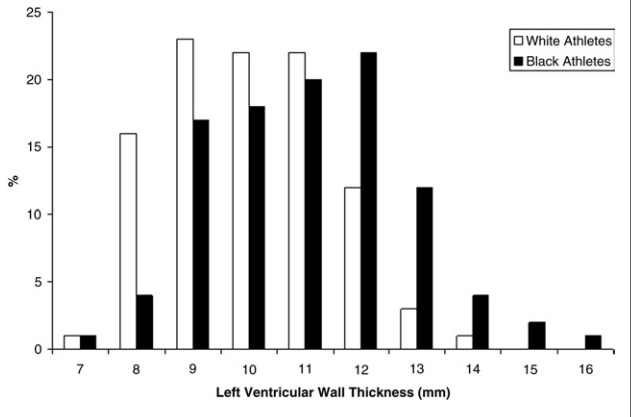

Figure 1 Bar Chart Showing the Distribution of Left Ventricular Wall Thickness in Black and White Athletes

A substantial minority (3%) of black athletes exhibited a left ventricular wall thickness ≥15 mm, compared with none of the white athletes.

Table 2 Demographics and Echocardiographic Parameters of 9 Black Athletes With LVH ≥15 mm

Athlete #	Age (yrs)	BSA (m^2)	Ancestral Origin	Sporting Discipline	LVW_d (mm)	$LVID_d$ (mm)	LA (mm)	E (m/s)	A (m/s)	E/A	E′ (m/s)	E/E′	Deep T-Wave Inversion
1	18	1.89	West African	Soccer	15	56	37	0.9	0.5	1.8	0.18	5	0
2	18	1.90	West African	Boxing	15	57	37	0.95	0.3	3.1	0.2	4.8	V_2–V_4
3	20	1.98	Afro-Caribbean	Boxing	15	57	39	1.1	0.4	2.8	0.23	4.8	V_1–V_3
4	23	2.04	West African	Sprinting	15	60	40	0.89	0.25	3.6	0.19	4.7	0
5	24	2.08	Afro-Caribbean	Basketball	15	60	40	1.2	0.5	2.4	0.22	5.5	0
6	26	2.09	West African	Basketball	15	62	39	1.4	0.3	4.6	0.25	5.6	V_1–V_4
7	19	2.15	West African	Boxing	16	57	41	0.96	0.4	2.4	0.2	4.8	V_3–V_4
8	24	2.24	West African	Basketball	16	65	42	0.9	0.3	3	0.19	4.7	0
9	26	2.32	Afro-Caribbean	Rugby	16	64	47	1.2	0.3	4	0.25	4.8	0

Abbreviations as in Table 1.

Left ventricular hypertrophy was predominantly seen in black athletes participating in boxing, basketball, soccer, and sprinting, most of whom were of West African ancestry. In contrast, relatively few white athletes of similar age and BSA, participating in the same sporting disciplines, exhibited LVH (Fig. 2). Black athletes participating in sprinting, boxing, and basketball exhibited a significantly greater magnitude of LVH compared with black athletes in other sporting disciplines (analysis of variance: $p < 0.01$). None of the long distance black runners, who were all East African, exhibited LVH.

Cardiac structure and function. The pattern of LVH in black and white athletes was homogeneous, with no athlete showing >2 mm difference in LV wall thickness measurements between contiguous segments. All athletes with LVH also exhibited an enlarged LV cavity size ranging between 55 and 66 mm and normal indexes of diastolic function on transmitral Doppler and TDI (Tables 1 and 2), and none displayed systolic anterior motion of the mitral valve and associated LV outflow obstruction.

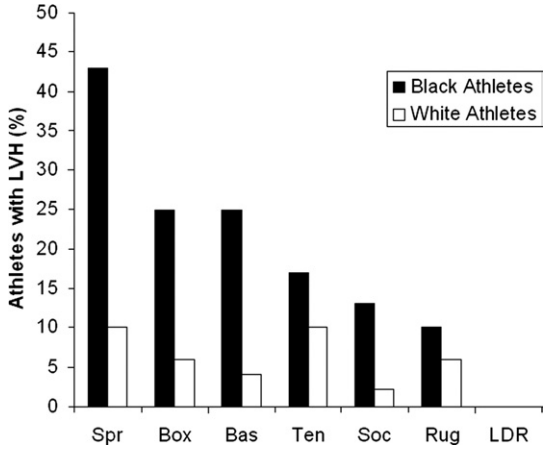

Figure 2 LVH in Relation to Sporting Disciplines in Black and White Athletes

More black athletes exhibited left ventricular hypertrophy (LVH) compared with white athletes in all 6 main sporting disciplines examined. Bas = basketball; Box = boxing; LDR = long distance running; Rug = rugby; Soc = soccer; Spr = sprinting; Ten = tennis.

ECG in black athletes with LVH. Black athletes with LVH exhibited a higher prevalence of voltage criteria for LVH compared with white athletes with LVH (37 [68%] vs. 5 [40%]; $p < 0.001$). Black athletes with LVH also displayed a higher prevalence of repolarization changes, specifically ST-segment elevation (46 [85%] vs. 7 [62%]; $p < 0.001$) and deep T-wave inversions (7 [12%] vs. none [0%]; $p < 0.0001$) compared with white athletes with LVH. Deep T-wave inversions (>0.2 mV) in black athletes were confined to leads V_1 to V_4 and present in 4 of the 9 athletes with substantial LVH ≥15 mm. Deep T-wave inversions were not an ECG manifestation in white athletes with LVH.

None of the athletes exhibited deep T-wave inversions in the inferior or lateral leads, baseline ST-segment depression, left atrial enlargement, pathologic Q waves, left bundle branch block, or leftward axis.

Determinants of LVH (all athletes). A multivariate linear model assessing the relation between LV wall thickness and several demographic variables showed an independent association between LV wall thickness and weight ($\beta = 0.008 \pm 0.002$; $p < 0.001$; 95% confidence interval [CI] 0.003 to 0.013) and African/Afro-Caribbean ethnicity (β 0.21 ± 0.03; $p < 0.001$; 95% CI 0.14 to 0.28).

Reliability of maximal LV wall thickness measurements in athletes. The averaged coefficients of variance for intraobserver and interobserver reliability for maximal LV wall thickness were 4.6% and 6.8%, respectively.

Exercise stress testing and 48-h ECG. Athletes with LVH underwent exercise stress testing and achieved >85% of the predicted target heart rate and adequate BP responses. None of the athletes exhibited ST-segment depression or arrhythmias during the exercise protocol. There were no differences in the mean peak systolic BP between black athletes and white athletes (189 ± 18.4 mm Hg vs. 183 ± 15.8 mm Hg).

None of the athletes with LVH showed episodes of nonsustained ventricular tachycardia or supraventricular tachyarrhythmias over a 24-h period.

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Discussion

Cardiac structure in highly-trained and nationally ranked black athletes has not been previously reported; therefore, the physiologic range of LV wall thickness measurements in this cohort is unknown. Sudden death from HCM in the U.S. is more common in black athletes compared with white athletes (5), underscoring the need for developing upper limits for LVH in black athletes to facilitate accurate differentiation between physiologic LVH and HCM.

Compared with the Italian Olympian study of 738 white male athletes in 1991 (5), only 300 black male athletes were studied, because black athletes constitute just 20% to 25% of the entire elite British athletic population. However, the study revealed that almost one-fifth of black athletes exhibited LVH compared with just 4% of white athletes. Furthermore, a significant minority (3%) of black athletes (but none of the white athletes) had substantial LVH (≥15 mm), which could have been consistent with morphologically mild HCM. These results are striking compared with the Italian Olympian athletes, in which only 0.08% of 738 men exhibited LVH ≥15 mm (1), indicating a racial predilection to developing LVH in response to the increased pre- and after-load associated with intensive exercise. A combination of genetic (18), endocrine, and hemodynamic factors (19) probably accounts for the increased LVH in black athletes. In the present study, basal and exercise-related BP responses in both groups of athletes did not differ and could not explain the increased magnitude of LVH in black athletes.

With the exception of sprinting, where black athletes dominate, we investigated sporting disciplines in which both black and white athletes participate in large numbers and excel equally, revealing that more black athletes than white athletes exhibited LVH in almost every sporting discipline examined. Disciplines such as cycling and rowing, which are associated with substantial LVH in white athletes, were not examined, because the lack of participation and, more importantly, failure to attain athletic excellence by black athletes in these disciplines would not enable direct comparisons.

Differentiating physiologic LVH from HCM in black athletes. The differentiation between physiologic LVH and HCM is crucial, because diagnostic errors have potentially grave consequences. In this regard, conclusions derived from LV wall thickness measurements in white athletes (1,3,4) may have resulted in a diagnosis of HCM and disqualification from competitive sport in 9 black athletes (3%) with LVH ≥15 mm. However, none of the 9 athletes exhibited other morphologic features suggestive of HCM. Specifically, none exhibited a nondilated LV cavity (4), enlarged left atrium >50 mm (20), LV obstruction (21), or evidence of impaired myocardial relaxation (14,22).

Indeed, all of the black athletes with LVH, including the 9 athletes with substantial LVH (Table 2), exhibited a large LV cavity (>55 mm) compared with individuals with HCM. In our experience, LV cavity size in HCM is <50 mm, even in affected individuals participating in regular sport against medical advice (23). An LV cavity >55 mm in HCM is due to progressive myocardial fibrosis and is associated with myocardial thinning, systolic dysfunction, and, importantly, reduced New York Heart Association functional class (24).

All black athletes with LVH also exhibited enhanced LV filling and normal left atrial pressures (E/E′ <7) on TDI, supporting physiologic adaptation rather than HCM. In contrast, TDI studies in HCM have convincingly identified LV filling abnormalities in gene-positive HCM individuals, even before the development of LVH (25).

Upright exercise testing and ambulatory ECG in athletes with LVH failed to identify abnormalities in BP responses to exercise (18) or nonsustained ventricular tachycardia (26), which constitute the broad spectrum of the HCM phenotype in a substantial number of affected individuals (27).

ECG in black athletes with LVH. In contrast to white athletes, 7 black athletes (12%) with LVH, including 4 with LVH ≥15 mm, displayed deep T-wave inversions in leads V_1 to V_4, a recognized ECG manifestation in HCM and arrhythmogenic right ventricular cardiomyopathy. In keeping with the recommendations of the 36th Bethesda guidelines (28) and the European Society of Sports Cardiology (29), we were compelled to investigate all 7 black athletes with cardiac magnetic resonance imaging (outside of the scope of the study) and failed to identify apical HCM, marked hypertrophy of the anterolateral wall (30), myocardial fibrosis, or characteristic right ventricular morphology-associated arrhythmogenic right ventricular cardiomyopathy in any athlete (31). In the absence of obvious pathology, we suspect these electrical anomalies (deep T-wave inversions in leads V_1 to V_4) in black athletes probably represent a normal spectrum of ECG changes in response to physical training (32), but we concede that long-term longitudinal studies are required to assess the precise significance of such repolarization changes in all athletes.

Diagnostic algorithm for differentiating physiologic LVH from HCM in black athletes with substantial LVH. The present study provides foundations for developing a pragmatic clinical algorithm for differentiating physiologic LVH from HCM in black male athletes age ≥16 years using readily available and independently interpretable cardiac investigations. As previously shown in white athletes (4), black athletes with physiologic LVH exhibit enlarged LV cavity >55 mm, a left atrial diameter ≥50 mm, and normal indexes of diastolic function on pulse Doppler and TDI in contrast to most individuals with HCM. Only black athletes age ≥16 years exhibited LVH (4); therefore, age is also pertinent when differentiating physiologic adaptation from HCM.

Although genetic testing is the most specific method of diagnosing HCM, the diverse genetic heterogeneity of

HCM and incomplete knowledge of all causal mutations do not currently allow a timely genetic diagnosis (>6 months) in athletes striving for honors or competing for team selection. Unfortunately, failure to identify an abnormality after screening for known mutations for HCM cannot currently be regarded as exclusion of disease.

Based on standard objective assessment we could not assign an LVH ≥15 mm in black athletes to be representative of inheritance of HCM-causing gene mutations in any athlete. Figures relating to the actual prevalence of HCM in the general population are also relevant in this regard, because the prevalence is only 0.2% (33) and certainly less in the athletic population (34); therefore, it is improbable on statistical grounds that 3% of all black athletes in the present study had HCM.

The substantial LVH identified in some athletes could have been attributed to the possible use of performance-enhancing drugs (35), but we were constrained on professional, ethical, and financial grounds from probing into drug abuse. However, all athletes had competed at the national level and were subject to random antidoping investigations.

Conclusions

Black athletes constitute a large proportion of athletes participating at the national level in the U.S. and United Kingdom. A substantial minority of black athletes also exhibit LVH ≥15 mm. We propose that in the absence of cardiac symptoms or a family history of HCM, an LV wall thickness ≥15 mm in black athletes may represent physiologic LVH when the LV cavity is enlarged and diastolic indexes are normal. Recommendations based on our observations should permit a more accurate evaluation of black athletes with LVH.

Acknowledgments

The authors thank Cardiac Risk in the Young for providing ECG and portable echocardiography machines for the study and Dr. Ameet Bakhai for statistical analysis of the data.

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